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7. AUTHOR(S) Genevieve Matowoski*; Emanuel Landau°; James Towascia*; Christiana Lazak*; Elizabeth A. Elliott*; William McEncone*; Richard Keating* Joseph Seifter+

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16. ABSTRACT

This interim report covers a study of excess mortality from cancer in the population residing near a chemical plant in Baltimore which had produced insecticides for 75 years.

Cancer deaths were examined for a five and nine year period surrounding census years to determine not only the absolute mortality but the changing trends.

Four index census tracts including the one containing the plant were selected based on the requirement that at least 50 percent of the area lay within a three-quarter mile radius of the plant. Comparison tracts were selected based on matching the index tracts by age, sex, and socio-economic status. Employee deaths were subtracted. The lung cancer death rate on an age adjusted basis had been found to be significantly higher in the census tract containing the plant. It had been rising rapidly since the mid-sixties. A study of the validation of hospital records and the pathology of cancer cases indicates that the excess risk of lung cancer represents a real risk in mortality and is not the result of local diagnostic and certification practices.

A corollary study of soil arsenic indicated highest levels in the tract with increased lung cancer mortality. The pattern of high soil levels appeared to be related to rail transport of the arsenical material.

Further research is required to determine whether other factors may play a role in the excess of lung cancer.

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LUNG CANCER/MORTALITY IN PROXIMITY TO A PESTICIDE PLANT

Genevieve M. Matanoski*

Emanuel Landau°

James Tonascia*

Christiana Lazar*

Elizabeth A. Elliott*

William McEnore*

Richard Keating*

Joseph Seifter+

Johns Hopkins University*
School of Hygiene and Public Health
Baltimore, Maryland

American Public Health Association°
Washington, D.C.

U.S. Environmental Protection Agency+
Office of Toxic Substances
Washington, D.C.

Project Officer
Charles Poole
Office of Toxic Substances
Washington, D.C.

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ABSTRACT

This interim report covers a study of excess mortality from cancer in the population residing near a chemical plant which had produced insecticides, herbicides and other arsenic products for three quarters of a century in the inner city of Baltimore.

A pilot study of three years mortality in the area compared to the remainder of the city indicated a three fold excess of lung cancer mortality for white males. As a result, cancer deaths were examined for a five and nine year period surrounding census years to determine not only the absolute mortality but the changing trends in approximately a twenty-year interval as part of the larger study.

Four index census tracts including the one containing the plant were selected based on the requirement that at least 50 percent of the area lay within a three-quarter mile radius of the plant. Comparison tracts were selected based on matching the index tracts by age, race, sex, median income, and for 1970 only, percent poverty level and percent head of household over age 65. Deaths among employees of the plant were obtained and subtracted to diminish the effect of possible direct occupational exposure to arsenic.

The lung cancer death rate on an age adjusted basis has been found to be significantly higher in the census tract containing the plant compared with other index tracts and controls. It has been rising rapidly since the mid-sixties. A study of the validation of hospital records and the pathology of cancer cases indicates that the excess risk of lung cancer represents a real risk in mortality and is not the result of local diagnostic and certification practices.

A corollary study of soil arsenic levels indicated that the recent contamination was less than previously experienced since the values at two inches below the surface were higher than at the surface in most areas. The pattern of high soil levels appeared to be related to rail transport of the arsenical material. High levels of arsenic in a generally northerly pattern were correlated with the location of the highest frequency of lung cancer cases as seen on the spot map.

Further research is required to determine whether smoking patterns, non-pesticide associated occupational exposure or residential history may play a role in the excess of lung cancer.

LUNG CANCER MORTALITY IN PROXIMITY TO A PESTICIDE PLANT

GENEVIEVE M. MATANOSKI, EMANUEL LANDAU, JAMES A. TONASCIA, CHRISTIANA LAZAR, ELIZABETH A. ELLIOTT, WILLIAM McENROE, RICHARD KEATING, AND JOSEPH SEIFTER

Introduction

Arsenic has long been recognized as a poison when ingested in large quantities by man, animals or plants. It is known that continued ingestion of high natural levels of arsenic in water or food will produce skin lesions including cancer (1,2). Consumption of arsenic as a therapeutic agent is also known to cause skin lesions (3).

The risk of inhalation of arsenic has not been as extensively investigated. Workers exposed to arsenic in the manufacture of pesticides have an increased risk of lung cancer and possibly lymphomas (4,5,6). Less is known about the chronic health effects in the general population exposed to arsenic in the air. Children around smelters may have high arsenic levels in nails and hair but it is not clear whether these observed indications of absorption of the agent can also produce long-term toxicity. Blot and Fraumeni (7) have

suggested that there may be an association between excessive lung cancer mortality and the existence of non-ferrous smelting industries in several counties in the U.S. It is possible that some by-product of this industry such as arsenic is associated with these carcinogenic effects.

The purpose of the current study was to determine whether there is an excess mortality from cancer in the population which resides near a chemical plant in the inner city of Baltimore and whether any observed excess can be associated with previous exposure to arsenic. The plant has produced insecticides, herbicides, and other arsenic products from 1897 until early 1976. In 1952, the original plant was torn down and a new one erected with better hygienic conditions for the workers. The plant produced arsenic acid, calcium and lead arsenate, Paris green (a cupric acetoarsenite), and sodium arsenite. All products were dried and packaged except sodium arsenite which was shipped as a liquid. Paris green was not produced after the early 1950's and no dry arsenicals were produced after 1973. Other pesticides such as chlorinated hydrocarbons and organophosphates were not produced at this facility but were made into formulations on-site since 1947. Further information about plant operations has not been sought.

There are several other industries which are currently

located in the area or have been manufacturing in that vicinity in the past. Adjacent to the chemical plant is a utilities company where natural gas was produced in the past through either a carburetted water gas or later an oil gas process. Currently, the company only stores natural gas in that area. Railroad yards are located to the northwest and southeast of the plant. There are other manufacturing industries in the area at this time and several have existed in the past within a one mile radius of the plant. These included a company which made rubber products, a small plant which produced fishing nets through a process which required dipping the product into organic materials and subsequent drying, and an abbatoir. Most of these operations have been defunct for the past ten years and little is known about their potential environmental hazards during the time they existed.

The pilot study, completed in April 1976, compared the three-year cancer mortality in this area to the rest of Baltimore. The results indicated that the census tract in which the plant was located had a higher all-cancer death rate than the rest of the city. 8/ The mortality for lung cancer specifically was three times higher in white males from that area than in men residing in similar areas throughout the city. The proposed expanded study of mortality was to include cancer deaths for each 5 year period surrounding a census year in order to determine not only the absolute

mortality but the changing trends over three census periods and to discover the possible errors in rates which resulted from deaths outside the city.

Method

The census tracts which were selected as having had possible environmental exposure to arsenic from the point source of the pesticide plant were defined empirically as those for which at least 50 percent of their area lay within a 3/4 mile radius of the plant. This distance was chosen so that large tracts which lay across the river and in which the majority of the population did not reside within a one mile radius of the plant would not be included. The four index census tracts which fit these criteria were 2303, 2302, 2404 and 2301. The tract in which the plant was located was 2303.

The comparison group of census tracts consisted of all tracts which matched the index ones on age distribution, race, sex, median income, and, for 1970 only, percent poverty level and percent head of household over age 65 years. Two separate matching groups were selected. Those designated as Match I were chosen for their similarity to the three tracts 2303, 2302, and 2404 which were alike especially in age and race distribution. The index tract 2301 had to be matched separately because its characteristics particularly in racial distribution, varied from the other

index tracts. By using this procedure for each census period, we found that different tracts matched in different census years. We also discovered that the boundaries of census tracts differed from one time to another. The most marked changes were between the characteristics of census tracts in 1950 and 1970, the extremes of time. However, we have utilized the same criteria for matching and have selected tracts in all time periods in the same way. Data from 1950-51 have not been analyzed for presentation at this time. For deaths in 1958 through 1962, the 1960 census tract data were used both for calculation of rates and establishment of matching tracts. For all other years, the 1970 census data were used for these purposes. Using separate matching procedures for each time period assured that for each period, the index and control tracts would be comparable for those variables on which we matched and mortality could be directly compared. However, they were not necessarily similar in matching variables over time. This did not concern us for race and sex which were analyzed separately but the age distribution of the populations in the index and control tracts had changed slightly. To account for this, some tables include an age-adjustment especially when rates are compared over time.

The initial matching criteria for the 1970-72 period were:

Age distribution ± 10% for each age

Race ± 15% expressed as % black

Sex ± 5% expressed as % male

Median Income ± $1,000

% below poverty level ± 10% in 1970 only

% head of household over 65 years ± 20% in 1970 only.

These figures for ranges in matching were arrived at empirically although a preliminary review of nearby similar tracts had suggested that these values would include the adjacent areas as controls. Thereafter, all tracts in the city which fell within these ranges were included as controls. We realized that using absolute income differences would represent a different extent of variation depending on the relative worth of the dollar in each decade. Therefore, we have expressed the original dollar difference of $1,000 as a percent of the median income in index tracts and this value of 12% was used to determine the differences in median income in index and control tracts in each census period. In 1960, this represented $700 and in 1950, $400 as maximum allowable differences in median income for matching tracts. This method of calculating relative income was based on the assumption of economic stability in the index area. Similar values would have been attained, however, if we had assumed a five percent annual income increase over the 10-year period. The matching tracts in 1970 and 1960 are shown in Tables 1 and 2. A total of 18 tracts matched the three

similar index tracts in the 1970 census and a total of 5 tracts match the 50-50 racial distribution tract in both the 1970 and 1960 censuses and these are designated as Match II. The variation in the number of matching tracts in the two calendar time periods is a reflection of the level of racial integration which has taken place in some of the middle income tracts over the past ten years. Those integrated tracts are no longer comparable in racial mix to the index tracts and so have dropped out in the 1970 matches. Also, housing in some central tracts was demolished during inner city reconstruction.

The comparabilities of index and control tracts are indicated in Tables 3 and 4 which display the characteristics of the tracts for four of the matching variables. As can be seen from these tables, the proportion of individuals aged 45 years and over residing in the index tracts has increased in the last decade. This is an indication of the stability of the population in the area near the plant. As a further marker of the permanence of the population, Table 5 indicates the mean percentage of the population occupying the household for seven or more years in 1960 and five plus years in 1970. These indicators would suggest that the Match I tracts and controls are very similar in population stability. Match II controls and index tract 2301 have more mobile populations. The tract 2303 which contains the plant has increased the

percentage of long-term residents from 55 in 1960 to 67 in 1970. It seems reasonable to assume that most of the 55 percent population which had been in tract 2303 since 1953 or earlier (residents seven or more years) have not all left the area in the next 5 years but constitute a high proportion of the 67 percent which were known to have occupied their homes from 1965 or earlier during the 1970 census (residents five or more years). It is highly probable, therefore, that over half of the population in that tract had lived in the same house for 17 or more years by the time of the 1970 census. For this reason, deaths occurring in residents during the period from 1968 on would probably represent the experience of long-term residents of the area. This explanation is only a presumption and could not be proven without a survey of the population to determine residence histories of households. We are hoping to be able to make this necessary survey.

All the previous tables have subdivided the control into four groups, the adjacent, the southern, the northern and the central controls. Figures 1 and 2 indicate the geographic distribution of all census tracts for both matching groups. The scattered distribution of the control tracts has placed them in areas which may also have had different risks. The adjacent tracts contiguous to the index ones may have had minimal exposure to the same agents in the major area.

Southern tracts are in heavy industrial areas as are the central tracts but the characteristics of the populations and their stability are different. The northern area consists of mainly residential dwellings with little industrial exposure.

Cancers were identified by examining all certificates of deaths which occurred within the city. The death was selected for study if cancer appeared as a cause listed anywhere on the death certificate. Deaths of city residents were selected from the total cancer list. This procedure would not include the deaths of city residents which occurred outside the city. These deaths had to be identified from another source which would have required more funding and time than originally proposed. If residents of certain areas of the city were more likely to have died at county hospitals than residents of other areas, we might have under-estimated the deaths to a greater extent in one area than the other and thus created a difference in mortality. In order to determine the extent of these differences we abstracted information on out-of-city deaths of city residents for the three years 1970-72. The proportional increase in deaths for the index tracts was 5 percent and for the control tracts 13 to 15 percent. This difference is not large enough to account for the variation in cancer rates observed.

Deaths were included only once regardless of number of cancer causes listed on a death certificate. The death was attributed to the underlying cancer or to the first cancer listed if there was an underlying cause of death other than cancer. Data from all census tracts were collected and analyzed in the same way so that comparisons between the matched tracts as well as with the total city are valid.

The original design of the study was altered to include more years around the 1970 census when the most rapid changes in cancer rates were observed. For the years 1966-67, the procedure described above was used since other data available for 1967 had missing deaths. However, for 1973-74 only the underlying cause of death as listed on a print-out has been used to identify cancers since death certificates were not readily available. Very few cancers, especially those in the lung, are missed by using this procedure.

The completeness of ascertainment of cancer deaths was determined by checking all records with a second abstractor. The omissions were 1.1 percent for the first abstraction. About half of the omissions were in cancers listed as underlying. The errors were corrected so that the overall missed cases after two reviews should be very small. The completeness of identification was also validated subsequently by matching our deaths against a tape of all deaths listed by underlying cause for all residents between 1968-72. Essentially no additional deaths were found by this method.

The coding of causes of death used two different classification schemes in the two periods with the period around 1960 using the 7th revision and the period of 1970, the 8th version. We examined the differences that these coding revisions had made in the classification of deaths and found that for some cancers the ICDA codes were essentially unchanged. We have included lung cancer not specified as primary within the total group of lung cancers. This procedure was indicated on the basis of coding practices and differences in the methods of recording causes of deaths in different census tracts. If "cancer of the lung" was recorded on a death certificate even though that was the only cause listed, it was usually coded as if it was not a primary lesion. The condition had to be specified as primary to be classified in that category. We found that the proportion of lung cancers, especially in the 7th revision, designated as primary differed by census tract and by time period even though the certificates appeared to be the same. We felt that the proportion of lung cancer deaths which were attributed to a primary lesion might depend on the precision with which local doctors filled out death certificates as well as coding practices, and thus, in turn, might vary by census tract and time. To combine the two classifications should not include the metastatic lesions but would include all types of lung cancers. For cancers such as cervix, breast, bladder, and prostate in which the code is entirely different

in the two coding revisions, the appropriate code for each time period was included separately.

Results

Mortality by Tracts

The crude rates for cancers at four specific sites-oral, pancreas, lung and prostate as well as for all cancers are presented for males in Tables 6 and 7. The former table includes data for the five-year period around the 1960 census and the latter table for a seven-year period around the 1970 census. The rates for individual tracts are examined separately since the tracts are located at different distances from the plant and therefore could have had a gradient in the populations exposure to pesticides. As can be seen, the risk for lung cancer and for all cancers is excessive in the period around the 1970 census for tract 2303 compared to any of the control groups. This is not consistently true in the earlier period. The weighted relative risk of lung cancer in white males from index tract 2303 as compared to north controls which had the highest control rate is 2.5 shown in Table 7 with a probability of .0005 as determined by the chi square calculated by the Woolf-Haldane method (9,10). The black males in tract 2301 also have a higher rate of lung cancer but this was not true for white males in the same tract. In the 1960 census period, although the lung cancer

rate is higher in white males in tract 2303 than the north and south controls, there is very little difference between the rates for all index tracts and for the adjacent and central controls. There are no differences in rates for males in tract 2301 and their control groups. Since the index and control tracts were matched for several variables no attempt was made to adjust for age in the initial calculations of mortality rates. Where differences have been noted in the first comparisons, adjusted rates will be presented subsequently.

For most control groups, the census tracts included in two census periods were not the same. However, some of the central area tracts from the 1960 and 1970 censuses were the same and rates are presented in Table 7A for those control and index tracts which are the same in the two periods. The rates for the central tracts are approximately the same for 1960 whether or not all tracts or the identical tracts from both censuses are included. These data would suggest that the rates for all of the areas were relatively similar in 1958-62 and that only 2303 had experienced a four-fold increase in lung cancer mortality and a two-fold increase in all cancers over the following 10-year period.

If we examine the comparable crude mortality rates for females in Tables 8 and 9, we can find no significant excess

risk of cancer at any site for census area 2303. In fact, the overall cancer rate appears somewhat low especially in the 1958-62 period. The mortality from breast cancer is slightly high in the early period and there are no deaths from cervical cancers. The lack of an observed increase in lung cancer in women in 2303 might be the result of a small population size. This will be discussed later in the report.

Mortality by Time

It was necessary to determine more precisely the time at which the increased mortality from lung cancer began in census tract 2303. Table 10 lists the crude average annual mortality rates for lung cancer in males for the two five-year periods around the census years and for two two-year periods before and after the 1968-72 rate. After the initial five-year period, 1958-62, when the mortality from lung cancer is similar for all index tracts and for the adjacent and central control tracts, the rate in tract 2303 shows only a slight rise in 1966-67. Throughout all of this early period, however, the lung cancer mortality is significantly higher in 2303 than in the northern controls. By 1968-72 the lung cancer mortality in males in 2303 has risen to about 4 times its original rate whereas the other areas shown have either a much less dramatic increase or no increase at all. The excess risk has persisted to 1973-74.

Both periods have significantly higher rates for 2303 as compared to northern controls. The rates for tract 2301 which lies north of the plant have also increased in this time period but the change is less impressive. All other index tracts have had a constant or decreasing rate.

Adjusted-rates

The age distribution of the index tract changed with time and these differences were reflected in similar changes in the control group. In order to have appropriate comparisons over time, the mortality rates for each cancer site and for all cancers have been adjusted using the method of standardized mortality ratios. The average annual Baltimore City mortality rates were calculated from all deaths in the 1968-74 period and these values were used as standards to adjust the mortality in each time period. As seen in Table 11, the mortality ratios for white males in tract 2303 were high for cancers of the lung, pancreas, stomach, prostate, oral cavity and all sites. The numbers of deaths except for lung and all sites combined were small but the pancreas cancer rate was still significantly higher than that for the city. White females in 2303 had an unremarkable overall cancer rate with excesses noted only for oral and rectal cancers of which only the latter ratio is significantly greater than unity. Since the colon cancer ratio is so low, it may be that an artifact of diagnosis locally may be classifying

colon cancers as rectal cancers erroneously. It seems unusual, however, that this should be happening only in females.

In Tables 12 and 13 and Figures 3 and 4 we examine the lung cancer mortality in two or three year time intervals. Using rates adjusted by the direct method to the 1970 Baltimore City population as a standard, we find that the death rate for this cancer has always been higher in males from tract 2303 than from most controls but that it has been rising rapidly. The total cancer rates have also shown higher values than among controls. A preliminary look at the lung cancer rates for 1950-51 indicated that the adjusted rates for that period were high for tract 2303 with a rate of 253 per 100,000 population as compared to rates ranging from 35.8 to 87.4 in other index tracts and controls.

Employees of an industry may live in close proximity to their place of work and could have accounted for an increased mortality in the census tract due to occupational exposure in the pesticide plant. With the cooperation of Dr. Abraham Lilienfeld of Johns Hopkins University and the company we reviewed lists of all employees to match with known deaths. Four employees were found among the cancer deaths in tract 2303 and two of these occurred among the lung cancers reported in 1969 and 1970. We felt it was important to determine how the index tract 2303 compared to all others in

the city and not just those tracts selected for study. The standardized mortality rations for lung cancer deaths in white males in every census tract in the city have been compared to the total white male lung cancer rate for Baltimore for the period 1970-72.

In table 12A deaths among employees for the index and adjacent tracts have been removed from the numerator and denominator of the rates. The resulting value is a minimum rate since we did not include any record of which employees were alive and residing in these same areas during the study period and thus, could not withdraw the living individuals from the denominator. The resulting rates still suggest that 2303 has a high mortality from lung cancer although 1968-69 rates now are lower than the northern controls.

Only tracts with SMR's of two or more have been included in Table 14 and Figure 5. These represent 14 tracts out of the total of 201 populated census tracts in the city. Tract 2303 has the highest ratio, the value being 4.21 times higher than the overall city rate. The nearest ratio of any other tract is 2.78.

The geographic distribution of cases was plotted on spot maps as shown in Figures 6 and 7 for two time periods. For both periods, lung cancer appears to be concentrated in an area about eight blocks wide and nine to twelve blocks long

lying to the north and east of the plant. The area encompasses all of tract 2303 and parts of 2302 and 2301. If one takes all of tracts 2303, 2301 and 2302 which lie within a 3/4 mile radius of the plant and calculates the proportion of lung cancers to all cancers in this area compared to the remaining census areas shown in Figure 6, the proportion is 44.4 percent near the plant and 16.8 percent in the outer areas. For Figure 7, the proportions show similar differences for 1973-74 as in the previous three years. For the area within the defined census tract and 3/4 miles from the plant, lung cancer represents 47.8 percent of the total cancers whereas in other areas it is only 33.3 percent. The northerly direction of this lung cancer excess is not compatible with the strong wind directions in that area. These winds arise in the northwest and west and should have carried contaminants to the east and southeast of the plant. The particles may have been moved by gentler winds and deposited nearby. (See Figure 8)

Discussion

An excess mortality from lung cancer has been demonstrated among men in a highly industrialized area of South Baltimore over the period from 1966 through 1974. Although the study was initiated to determine whether there was an environmental impact from exposure to arsenic, the current information does not provide evidence of a direct association.

There are some questions which arise with regard to the data. Why did the excess risk appear primarily in the late 60's and early 70's when the plant had existed and produced arsenical products since the early 1900's? The discrepancy could indicate that the plant did not account for the excess but some other local industry or occupational group accounts for the excess. It is also possible that the men in the area had a higher frequency of smoking and smoked more cigarettes than did the populations in the rest of the city. Moreover, it is possible that selective out-migration of younger, healthier males created a higher risk among the remaining population. Both of the latter explanations could be determined by a survey.

The sudden rise in lung cancer might be related to the destruction of the old plant in 1952. Such an undertaking could have spread dust diffusely throughout the community. Under these circumstances, we must ask why the concentration of lung cancer in the area does not coincide with the assumed wind spread of particles. It is necessary to examine further the mortality in the 1950 period to determine whether an excess existed at that time before the destruction of the old plant. It would be interesting to see if the appearance of the excess risk of lung cancer in the community coincided with that found in the workers within the pesticide plant. If we presume that arsenic may not be causing the excess, then it would be necessary to examine the mortality experience of workers in other industries in the area,

especially the natural gas plant, to see if they have an excess lung cancer mortality. In almost all cases, workers within an industry should have higher exposure and a greater risk of disease than the general public. It is necessary to investigate whether the increase in lung cancer can be related to a change in production or methods of operation of any of the businesses. For example, differences in handling arsenic, changes in formulation of pesticides or the conversion of the gas plant from carburetted water gas to oil gas production could have created variations in level or type of pollution.

The fact that the excess lung cancer mortality has occurred only in men raises the question as to whether an environmental factor or differences in smoking characteristics or occupation have caused the increased death rate in tract 2303. It is possible that smoking and an environmental pollutant are required to produce the excess of cancer. The rates in older women then could be lower because they did not smoke and the possible syngergistic effect of cigarettes and the environmental factor were not observed. Additional research to ascertain the rate of cigarette smoking in the reported excess lung cancer is clearly necessary.

Supplementary Studies

In the meantime, further studies have been done to determine whether the excess cancers might be associated with exposure

to arsenic. Soil samples have been collected in the community as a proxy measure of the presence of arsenic in the air at a time when air sampling data were not available. Validation of medical histories and the cell type of the cancers were studied to determine whether these factors differed in individuals whose lesions might have been caused by arsenic because of residence in the index area. These factors are fully covered in the Appendices. However, let us look at the soil sampling study and the validation of hospital records and pathology of cancer cases in more detail.

Soil Sampling

As noted above, the purpose of the sampling of soil in the area near the pesticide plant was to try to determine whether arsenic had escaped into the community and whether the soil level of this substance at varying distances from the plant would suggest the direction of air spread of the agent. A review of the experience of others in using a soil level as a proxy measure of air values had demonstrated the success of such a procedure. Landrigan et al. (11) had tested lead and other metals including arsenic in an area around a smelter. They had demonstrated that lead levels in the air fell rapidly to a distance of 2-3 Km. from the plant and more gradually thereafter until reaching background levels at 4-5 Km. The dust sampling also indicated higher levels closer to the plant except where there was shielding

by mountains. Soil tests done in the same area indicated a similar rapid dropoff in levels to 2-3 Km. but the values remained elevated to longer distances of 10 Km. The tests were run at the surface and at 2.5, 5.0 and 7.5 cm. depths with the highest lead levels being consistently found at the surface in this situation with continuous contamination. The environmental levels correlated well with blood lead levels in children. Milham and Strong (12) tested arsenic levels in urine of children living near a smelter and found a relationship with household dust. Dorn et al. (13) studied the dustfall, soil, and vegetative levels of lead and other metals originating from a lead smelter and correlated these with levels in blood and milk of nearby cattle. The results indicated that there were differences in the suspended particulate level for metals by season with the winter having the highest and the spring the lowest level and that these levels correlated well with dustfall, with cattle body burdens, and with the periods of greatest wind velocity. Using these studies as models we presumed that arsenic might act in a similar manner to lead and that soil levels could represent previous air-borne exposures even though the chemical plant did not have the tall stacks usually associated with smelters and the surrounding geographic characteristics of a city might alter the relationships reported from a relatively open and rural type of environment.

The validity of our assumptions were investigated by contacting

local consultants concerning the sampling of soil for arsenic, the characteristics of Maryland soils, the possible changes in arsenic with weathering in soil, and the influences of local topography on deposition of arsenic. We consulted individuals from the U.S. Geological Survey Unit and the Environment and Geography Department at the Homewood Campus as well as those from the Environmental Health Department at the Medical Campus of the Johns Hopkins University. Individuals from the University of Maryland and the Maryland State Department of Agriculture also provided information and references. The consensus of opinion was that few tests for arsenic had been done in Maryland but it was felt that, unless deliberate additions of arsenic-containing materials had occurred, very little of the metal should be present in the soil naturally. Dr. Cady and others warned about collecting samples under trees and in areas where wash-off could occur since these situations could either reduce the amount of dustfall from air-borne particles or dilute the level of penetration into the soil.

The method of selecting samples is covered in Appendix I.

Results

The selection sites are shown in Figure 9 and the levels of arsenic in Table 15. If we examine only the 101 samples taken in the first set we can see that, in general, arsenic

levels were highest where lung cancer mortality was also highest. The mean arsenic level from 20 sample sites in tract 2303 was 63 ppm of arsenic. Even the omission of samples from the park adjacent to the plant only reduced the mean arsenic level to 38 ppm. Tracts 2301 and 2404 had means of 6 ppm and 2302 a mean of 4 ppm based on only 2 to 4 sample sites. At most sites, the arsenic level was highest at two inches. This suggested that recent contamination had been less than in the past. The highest and mean levels at various sites are shown in the maps, Figures 10 and 11. If we examine the samples omitting sites 133, which were taken during the 1977 spring period, we find the highest levels in the park at the northern fence in an area which was adjacent to the property line of the plant. It was also in this area that one inch deep samples sometimes had the highest value suggesting a more recent dustfall containing arsenic. All sites in the park had high levels except for an area which had been turned over and resodded and in which low arsenic levels were present. The one inch levels near the fence were as high as 695 and 226 ppm whereas at the opposite side of the park the values were only 29 to 97 ppm at one inch but as high as 46 to 161 ppm at two inches deep. From the soil levels of the original samples, the data indicated high levels near the plant to the east and north and south in the park within a 3/8 mile radius of the plant. It was also apparent that higher levels were found in a northerly direction along the railroad lines and these levels had not

dropped as far north as was tested in the first set of samples. It was decided to collect additional samples to the north along the railroad lines to determine the point at which arsenic levels dropped to control values. Samples were also collected to the east in the Port Covington yards and along a spur to indicate levels associated with the presence of coal and railroads.

The second set of samples include sites 1-33 listed as Spring 1977. These samples, in general, had moderately high levels of arsenic at three sites and there was no pattern of a gradient in the levels at the sites most distant from the plant. The level in the railroad yard sample was high with a mean of 52.3 ppm. This value was higher than the other sites east of the plant. Two sites to the north had unusually high levels with means of 59.7 and 92.7 and highest values of 144 and 173. These values do not follow a pattern of decreasing levels at further distance from the plant.

Discussion

The initial samples indicated that arsenic had spread into the general environment especially into the park adjacent to the plant. The gradient of level by depth at each site suggested that recent contamination had been less than previously experienced pollution since the highest value most frequently occurred at two inches and not near the

surface. The levels at four inches were usually lower than at the shallower depths but still correlated well with the values at the surface. We felt the values at two inches suggested generally long-term contamination of the area although this could not be proven since we found no information on the time required for percolation of arsenic through soil. There was a gradient both to the north and the east in earlier samples which suggested that wind currents toward the east might have carried the material about 1/4 mile but the spread was further in a northerly direction. It was also apparent that the heaviest contamination was in the park near the plant. It was impossible to detect high levels in soil directly adjacent to areas where lung cancers occurred although the higher levels of arsenic in a generally northerly pattern correlated with the location of the highest frequency of lung cancers as seen on the spot map. It was proposed that the pattern of high levels might be related to rail tansport of the material since an active rail line did connect to the company. Fugitive emissions from this transportation route might have accounted for the erratic distribution of arsenic by gradient of level.

The further samples collected at sites in northerly and easterly directions along railroad lines and into the railroad yards created some questions about the spread of arsenic from the plant. The levels in the Port Covington yard were high with values of 41 to 60 ppm arsenic at a site

approximately 10 feet from the railroad tracks in a flat area with no run-off. At first we thought these values could be explained by the presence of coal dust in the soil. Mr. Janus, chief of coal sampling and inspection from the Bureau of Mines provided information on the components of the Pittsburgh and Upper Freeport seam coals which are the types found in the Baltimore years. As reported by Kessler, Sharkey and Friedel (14), neither the ash nor the dust has more than 16 ppm of arsenic, a value much lower than those observed.

The second possible explanation for the presence of arsenic in the yards was that pesticides were transported there in open cars. That would not be possible under current conditions since the yard handles primarily containerized freight except for coal. There are no records of what materials it handled previously and how they were transported. It seems unlikely that arsenic in open cars would have contaminated the yards without also leaving material along the tracks nearer the plant. No sites were sampled along those tracks and, thus, further sampling in these areas is needed.

The third possibility was that herbicides had been used around the yards and along railroad lines to control vegetation. There are no records of any herbicide use in the area. The high levels at all depths would suggest repeated high concentrations over long periods as one would expect with

herbicide treatments. However, the levels along a smaller track to the north shows no signs of high levels of this magnitude. If herbicides are the explanation there must have been selective use of the product on different railroad beds.

High levels are seen north of the natural gas plant and there is no gradient by distance from the plant as expected. The sites sampled in these areas were at least 30 feet from the railroad track, a distance which is probably too far to be contaminated by herbicides although there could be distant run-off from applications to the bed. We could postulate that material had been moved from the plant in a northerly direction with varying degrees of contamination of soil depending on track slope or other factors but further information is needed especially to explain the lack of a gradient.

Additional consultation was sought from Dr. Woolson at the Pesticide Degradation Laboratory in Beltsville. Except for the suggestion that sodium arsenite or perhaps the methanearsonates used on the railroad beds might play a role, he had no other recommendations. Additional information could be sought from the company about methods of transportation if further sampling is anticipated so that more appropriate sites can be selected for study.

There are many other possible steps which could be taken. Information is needed on the time of contamination which would be represented by the presence of arsenic at a depth of 2 inches. This might require simulated conditions if there are not data from other sources. We should also examine the possibility of testing the sediment in the surrounding waters for arsenic unless pollution from multiple sources represents too much of a problem. Sites from the second sampling set could be repeated using closely adjacent spots to see if the discrepant levels represented some type of local contamination. Many of the areas in that vicinity have had recent changes in topography, however, due to construction of a bridge in that neighborhood which may have eliminated some of the original sites.

Validation of hospital records and cancer pathology

This phase of the study of the possible association of mortality with exposure to pesticides included a review of hospital records of patients. (Appendix II) The purposes of this phase were to determine the accuracy of death certification of cancers in Baltimore; to identify any possible differences in diagnosis by area; to determine any variation in the pathological characteristics of cancers especially those of the lung in the index census region as compared to others; and to investigate any possible differences in personal characteristics as described in hospital records. The

investigation would focus specifically on possible identification of unusual cell types of lung cancer in the presumed arsenic-exposed group and the presence of histories of known arsenic-associated diseases in the patients dying of lung cancer in the index area.

Results

The causes of death were grouped into two time periods which represented the use of the 7th and 8th ICDA codes and grouped into causes as listed on the certificate by the first two digits of the code. These causes were then compared to the first four medical conditions or diagnoses as noted on the hospital records. The individuals in whom the records did correspond within the first two digits for lung cancer and total cancer only are noted on Table 16. It is apparent that 15.0 to 18.5 percent of cancers identified by death certificate do not have the same diagnosis as listed among the first four diagnoses on hospital records.

The problem of validation of death certificate information was reviewed further by a physician who examined the data on the abstract forms. As indicated in Table 7, there was complete agreement in diagnoses to four digits in the ICDA code in only 75.0 percent of the cancer deaths. If classification to three digits only is used we will correctly verify 80.7 percent of the cases listed on the certificate. In 1.8

percent of case metastatic lesions were identified on the death certificate as underlying and in another 5.5 percent multiple cancers were listed on the certificate and the primary site varied from that listed on the hospital record. There was no cancer diagnosis listed anywhere on the hospital record for 2.7 percent of deaths. A further examination of the method of diagnosis of cases was attempted in order to demonstrate whether differences in the methods of diagnosis might have changed the accuracy of death certification. Data from autopsy or histological examination of tissue was used for the diagnosis for 82.9 percent of the cases with complete agreement in records and 88.9 percent of cases where the agreement was less than perfect. Therefore, the reliability of the hospital diagnosis as determined from the method by which the cancer was identified did not select those cases which were inappropriately classified on the death certificate.

The method by which the diagnosis of cancer was made differed only slightly in the larger hospitals with 69.7 to 91.4 percent of cases diagnosed by autopsy or histology. An examination of the differences in diagnosis by census tract has not been completed, but it is unlikely that there will be variations in diagnosis since we have included all hospitals used by individuals from the index area in the above evaluation. An examination of the hospital records for possible arsenic-associated symptoms included

gastrointestinal signs, skin lesions, Mee's lines on nails, neurological or neuromuscular symptoms, cardiovascular disease, stroke and asthma. Only respiratory symptoms were slightly higher in the index tract but since there were so many records in which there was no comment about these symptoms, it is difficult to interpret the small variation.

We also sought information on diseases for which arsenic might have been used as a treatment, such as syphilis, trypanosomiasis and amebiasis and the results indicated no higher frequency of these conditions among residents of index tracts. As might be expected, most of these diseases were rarely recorded for deaths in any area.

Both smoking and drinking histories were abstracted from hospital records. Drinking was rarely recorded and smoking histories were frequently missing. Table 18 indicates the smoking characteristics of lung cancer deaths in index and control tracts as determined from the hospital record. For 46.6 percent of the patients, the smoking histories are unknown. Despite that fact, we have attempted to compare the smoking levels in index versus the control tracts. The percent of smokers is slightly higher in the index tracts but the difference does not appear impressive. If one includes only those charts with a recorded history, almost all cases are positive for smoking in both index and control tracts.

The original hypothesis was that if arsenic had caused the lung cancers, the cell type of lesions from the index tract might differ compared to other areas with an expected predominance of small cell or oat cell tumors in the exposed tracts. The data in Table 19 would indicate that the cell types differ very little from index to control tracts.

Discussion

Despite the suspicion that the characteristics of individuals in tract 2303 might differ from control tracts because of exposure to arsenic, there were no indications from the data that they had an increased risk of arsenic-associated diseases. They also did not have differences in smoking histories or in the frequency of the cell types of lung cancers. If we could have shown an excess risk of smoking in tract 2303, we might have explained the increased mortality from lung cancer on the basis of differences in cigarette consumption. If we could have shown an increased frequency in specific cell types among the lung cancers from tract 2303 as compared to controls, we could have suggested that exposure to a pollutant other than cigarette smoke was the probable cause of the increased lung cancer mortality. Neither situation occurred. Actually, the number of charts which had adequate records to answer questions about smoking were very few. Only a survey would provide the data which are needed for such a review.

It is apparent from the data that the classification of deaths from death certificates are often in error compared to the original hospital record. However, if we adhere to major categories of cancer, most of the records do correspond. For about three percent of the records, there is no clue as to how the diagnoses were determined. It is possible that these were the wrong records despite the multiple methods of identification. It is also possible that a history of cancer was missed in the abstracting. It is safe to say that death certificate information for Baltimore City is usually accurate in identifying a cancer if the first two digits of the classification only are considered. Therefore, the excess risk of lung cancer identified in tract 2303 is a real increase in mortality and not the result of local diagnostic and certification practices.

Conclusion:

The data from a study of cancer mortality in an industralized area of Baltimore where an arsenical-producing pesticide plant was located indicated a significant excess risk of death from lung cancer and all cancers among males. The risk has increased in the period around the 1970 census as compared to previous time intervals. No risk of lung cancer was found in females nor in residents of census tracts located over one-half mile from the plant. The excess was not due to occupational exposure to pesticide manufacture.

Soil levels of arsenic were higher in the areas where the mortality from lung cancer was also higher. The arsenic levels appeared to be increased in the area around the north-bound railroad line. Although there was a gradient of reduced arsenic levels with increased distance from the plant, it was not a perfect correlation for all sites along the railroad, possibly because of erratic fugitive emissions from a transport route.

The excess lung cancer mortality in the local area could not be attributed to differences in diagnostic practices between hospitals in the city. Smoking histories in hospital records were very incomplete but did not indicate differences in cigarette use in index and control census tracts. No symptoms of arsenic-related disease could be identified on the hospital records. The frequencies of specific cell types among the lung cancers in the index tracts and control tracts were similar.

Further research is required to determine whether smoking patterns, non-pesticide associated occupational exposure or residential history may play a role in the excess of lung cancer.

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Table 1.

Designation of 1970 Census Tracts

	Index Tracts	Adjacents Controls	South Controls	Central Controls	North Controls
Match I	2302	2401	2005	101	1308.02
	2303*	2402	2502.05	104	2604.01
	2404	2403	2503.03	105	2702
			2504.01	601	
			2504.02	602	
			2505	2608	
Match II	2301	2101		703	905
		2201			1203

* Plant located in this tract.

Table 2.

Designation of 1960 Census Tracts

	Index Tracts	Adjacent Controls	South Controls	Central Controls		North Controls
Match I	2302	2401	1903	101	703	904
	2303	2402	1904	102	2603	905
	2404	2403	2003	103	2607	906
			2005	104	2608	1305
			2008	105	2609	1306
			2504	201	2610	1308
			2505	202	2611	1512
			2503B	203	2604A	1513
			2502C	601	2606A	2716
				602	2604B	2718
				701		2801A
				702		
Match II	2301	2201		604		1104
						1205
						1401

Table 3. Characteristics of Index and Control Census Tracts from 1970 Census for Selected Baltimore Areas

Characteristics	Match I							Match II			
	2303	2302	2404	Adjacent Controls	South Controls	Central Controls	North Controls	2301	Adjacent Controls	Central Controls	North Controls
Total population	1703	3395	3697	9347	31,174	20,378	12,964	3560	5606	3180	8139
Range between tracts				2872-3358	3143-6893	2125-4855	2316-5969		1935-3671		2952-5187
% < 5 yrs	8	8	8	6-8	6-12	6-8	6-10	10	8	10	9
% 5-19 yrs	29	28	28	25-26	24-30	23-28	23-26	30	28-30	30	29-33
% 20-44 yrs	26	28	29	28-29	27-39	26-32	26-35	26	25-26	26	32-34
% 45-64 yrs	28	24	25	23-38	14-28	25-33	22-27	23	25-26	23	17-19
% 65+ yrs	9	11	10	10-16	4-13	12-15	6-17	10	12-13	12	8-10
% Male	50	50	48	48-49	47-49	46-50	47-49	49	48-54	48	47-48
% Black	-	0	-	0-2	0-2	0-2	0-4	50	50-52	47	39-64
Median Income (Family)	8400	9200	8400	8100-9300	7500-9000	7500-9300	8800-10000	6500	5800-6000	6800	7000-7300

Table 4. Characteristics of Index and Control Census Tracts from 1960 Census for Selected Baltimore Areas

Characteristics	Match I							Match II			
	2303	2302	2404	Adjacent Controls	South Controls	Central Controls	North Controls	2301	Adjacent Controls	Central Controls	North Controls
Total population	2135	3924	4204	10,529	49,997	108,839	53,874	4100	3253	4367	15,133
Range between tracts				3178-3682	2562-12163	2548-17511	1762-6264				3134-6843
% <5 yrs	10	10	10	10-11	8-12	7-15	6-11	13	11	16	12-14
% 5-19 yrs	29	26	27	27	21-28	20-33	17-27	28	24	37	20-23
% 20-44 yrs	31	31	31	31-33	28-37	23-38	25-35	30	32	25	33-39
% 45-64 yrs	22	22	22	21-24	17-27	15-31	21-29	22	25	17	18-20
% 65+yrs	8	10	9	9	5-16	2-14	8-22	9	8	6	8-12
% Male	51	48	50	49-50	47-52	47-53	44-49	49	55	50	46-51
% Black	1	1	0	0-1	0-8	0-13	1-12	60	49	51	47-71
Median Income (Family)	5600	5100	5600	5500-6200	4800-6200	4800-6300	4900-6300	3800	3600	3300	3300-4100

Table 5. Duration of Residence in Household

	1960*		1970*	
Match I	Mean	Range	Mean	Range
Index	58	54-65	64	55-69
Adjacent Controls	62	55-70	67	55-76
South Controls	54	43-65	62	57-72
Central Controls	57	18-68	63	50-69
North Controls	56	49-61	64	60-67
Match II				
Index	46	46	59	59
Adjacent Controls	44	44	60	54-67
Central Controls	34	34	49	49
North Controls	28	22-32	48	44-53

* 1960 % living in same house 7+ years
1970 % living in same 5+ years

Table 6

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1958-62

Males

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	2	21.1	1	9.5	5	19.3	10	8.3	20	7.6	11	8.9
Pancreas	1	18.5	0	-	2	19.0	3	11.6	8	6.6	31	11.8	18	14.6
Lung	4	74.1	12	126.8	8	75.9	23	88.8	79	65.4	193	73.6	60	48.8
Prostate	2	37.1	2	21.1	3	28.4	3	11.6	19	15.7	43	16.4	23	18.7
All Cancer	11	203.9	26	274.8	32	303.5	56	216.1	244	202.1	615	234.6	293	238.2

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	1	21.8	0	-	0	-	0	-	3	18.4	0	-
Pancreas	0	-	1	16.8	0	-	1	22.9	1	17.7	1	18.9	2	12.2	0	-
Lung	3	75.2	2	33.5	2	43.6	1	22.9	5	88.7	6	113.6	12	73.4	8	39.3
Prostate	1	25.1	2	33.5	1	21.8	1	22.9	0	-	0	-	5	30.6	3	14.7
All Cancer	10	250.6	16	268.2	8	174.3	12	274.9	14	248.2	17	322.0	43	263.2	39	191.6

Table 7

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1968-74

Males

Match I

Census Tracts

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	2	33.9	0	-	0	-	4	12.7	7	6.8	6	9.0	5	11.7
Pancreas	2	33.9	0	-	1	8.0	3	9.6	9	8.8	9	13.4	3	7.0
Lung	18	305.0	12	103.3	12	96.1	24	76.4	111	108.3	60	89.6	48	112.4
Prostate	1	16.9	1	8.6	2	16.0	4	12.7	15	14.6	25	37.3	7	16.4
All Cancer	33	559.2	26	223.8	24	192.2	86	273.9	254	247.9	206	307.7	110	257.7

Match II

	2301				Adjacent				Central				North			
	White		Black		White		Black		White		Black		White		Black	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	1	16.9	2	20.5	2	20.3	1	17.5	0	-	0	-	1	6.9
Pancreas	0	-	3	50.7	0	-	3	30.5	2	35.1	0	-	0	-	1	6.9
Lung	7	113.3	13	219.5	13	133.1	16	162.6	4	70.1	5	102.8	15	124.5	6	41.3
Prostate	1	16.2	3	50.7	4	41.0	4	40.6	1	17.5	2	41.1	3	24.9	3	20.7
All Cancer	17	275.0	27	455.9	34	348.2	42	426.7	17	298.0	13	267.2	41	340.3	28	192.9

Table 7A

Crude Lung and All Cancer Deaths Rates per 100,000
White Males in Census Tracts the Same in 1960 and 1970

	Census Tracts				
	2303	2302	2404	Adjacent	Central
Lung Cancer					
1958-62	74.1	126.8	75.9	88.8	77.7
1968-74	305.0	103.3	96.1	76.4	89.6
All Cancer					
1958-62	203.9	274.8	303.5	216.1	241.5
1968-74	559.2	223.8	192.2	273.9	307.7

Table 8

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1958-62

Females

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	0	-	0	-	2	1.6	2	0.7	4	2.9
Pancreas	0	-	2	19.9	0	-	0	-	8	6.4	23	8.4	12	8.8
Lung	0	-	0	-	0	-	6	22.6	8	6.4	18	6.5	15	11.0
Breast	3	57.5	2	19.9	3	28.7	8	30.2	28	22.5	81	29.4	51	37.5
Cervic	0	-	1	10.0	1	9.6	3	11.3	15	12.0	34	12.4	12	8.8
All Cancer	6	115.1	17	169.2	13	124.4	43	162.3	186	149.3	460	167.2	263	193.3

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	1	27.4	0	-	0	-	0	-	0	-	0	-
Pancreas	0	-	0	-	1	27.4	1	27.3	0	-	0	-	1	5.8	0	-
Lung	0	-	1	15.8	0	-	0	-	1	19.7	0	-	1	5.8	0	-
Breast	3	71.4	2	31.5	3	82.2	1	27.3	2	39.5	1	17.1	8	46.6	4	18.3
Cervix	1	23.8	1	15.8	1	27.4	1	27.3	2	39.5	1	17.1	5	29.1	3	13.8
All Cancer	6	142.9	10	157.6	10	274.0	8	218.6	9	177.7	6	102.6	41	238.9	21	96.3

Table 9

Average Annual Crude Death Rate per 100,000 for Index and Control Census Tracts

Deaths 1968-74

Females

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	2	33.3	0	-	0	-	4	12.1	3	2.7	3	4.1	2	4.3
Pancreas	0	-	2	16.8	2	15.0	1	3.0	11	9.9	10	13.6	1	2.2
Lung	1	16.6	3	25.2	1	7.5	7	21.1	16	14.4	18	24.5	9	19.5
Breast	1	16.6	1	8.4	2	15.0	8	24.1	25	22.5	29	39.5	9	19.5
Cervix	0	-	3	25.2	0	-	3	9.1	6	5.4	5	6.8	0	-
All Cancer	9	149.9	14	117.8	17	127.3	56	169.0	165	148.7	175	238.2	78	169.2

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
Oral	0	-	0	-	0	-	1	9.7	0	-	0	-	0	-	0	-
Pancreas	1	16.4	0	-	1	10.9	2	19.3	1	16.7	0	-	3	23.0	0	-
Lung	2	32.8	1	15.4	2	21.9	7	67.6	1	16.7	1	18.1	3	23.0	0	-
Breast	5	82.0	3	46.2	6	65.7	4	38.6	2	33.4	0	-	8	61.4	3	18.1
Cervix	0	-	2	30.8	3	32.8	1	9.7	0	-	1	18.1	2	15.4	1	6.0
All Cancer	15	246.0	18	277.1	26	284.6	25	241.5	14	233.6	5	90.5	34	261.1	16	96.3

Table 10

Average Annual Crude Rates per 100,000

Male Lung Cancer

Match I

Years	2303	2302	2404	Adjacent	South	Central	North
1958-62	74.1*	126.8	75.9	88.8	65.4	73.6	48.8
1966-67	177.9±	180.7	84.1	111.5	54.6	151.6	82.0
1968-72	284.7*	120.5	100.9	66.9	110.7	85.7	114.8
1973-74	355.9*	60.2	84.1	100.3	102.5	99.3	106.6

Significance tests * $p < .05$
± $.10 > p > .05$: Compared to Northern Controls.
Woolf-Haldane relative risk analysis.

Match II

	2301		Adjacent		Central		North	
Years	White	Black*	White	Black	White	Black	White	Black
1958-62	75.2	33.5	43.6	22.9	88.7	132.6	73.4	39.3
1966-67	56.6	-	358.4	248.9	61.3	71.9	145.3	-
1968-72	90.6	260.0	129.0	184.9	98.2	115.1	93.0	38.6
1973-74	169.9	118.2	143.4	106.7	-	71.9	203.4	48.2

* 1958-62 rates are for nonwhites.

Table 11

Time-Adjusted SMRs Based on Average Annual Baltimore City Rates*

Deaths 1958-1962 and 1966-1974

Match I White Males

	2303		2302		2404		Adjacent Controls		South Controls		Central Controls		North Controls	
Cause	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR
Oral	3	2.45	2	0.88	1	0.43	9	1.43	18	0.88	29	0.91	16	0.89
Stomach	2	1.60	3	1.18	4	1.59	10	1.44	23	0.98	60	1.40	17	0.70
Colon	3	1.22	9	1.74	6	1.21	9	0.62	43	0.94	67	0.91	38	0.90
Rectum	1	1.09	3	1.62	4	2.19	6	1.18	14	0.85	34	1.25	15	0.97
Pancreas	5	4.15	0	-	3	1.31	7	1.09	21	1.01	43	1.27	21	1.10
Lung	25	2.74	30	1.72	23	1.31	57	1.19	206	1.35	282	1.21	118	0.93
Prostate	3	1.59	3	0.68	5	1.26	7	0.56	36	0.93	74	1.15	31	0.79
Bladder	0	-	3	1.38	2	0.97	5	0.85	20	1.03	37	1.11	28	1.45
Lymphomas	2	0.88	4	0.89	2	0.44	10	0.80	26	0.62	65	1.02	37	1.06
All Cancer	54	1.94	67	1.21	63	1.15	168	1.10	543	1.10	891	1.13	423	0.96

Match I White Females

	2303		2302		2404		Adjacent Controls		South Controls		Central Controls		North Controls	
Cause	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR	obs	SMR
Oral	2	6.31	0	-	1	1.40	4	2.06	5	0.84	5	0.57	6	1.14
Stomach	1	1.32	3	1.74	0	-	5	0.96	14	0.89	37	1.30	18	1.01
Colon	1	0.45	7	1.36	5	0.94	13	0.83	41	0.89	81	1.01	40	0.82
Rectum	4	6.12	0	-	2	1.32	3	0.69	14	1.08	33	1.57	13	1.03
Pancreas	0	-	4	2.32	2	1.13	1	0.19	20	1.34	38	1.59	13	0.90
Lung	1	0.71	4	1.34	1	0.31	15	1.70	26	0.99	37	1.05	23	1.14
Breast	5	1.29	5	0.61	6	0.68	17	0.70	58	0.75	119	0.94	61	0.82
Cervix	0	-	5	2.30	3	1.27	6	0.97	25	1.13	43	1.14	13	0.61
Bladder	0	-	0	-	0	-	0	-	10	1.33	15	1.15	6	0.74
Lymphomas	1	0.65	4	1.19	3	0.86	6	0.61	36	1.22	52	1.05	24	0.83
All Cancer	18	0.96	41	1.00	39	0.91	109	0.90	390	1.05	682	1.10	350	0.96

* Average annual Baltimore City rates (based on deaths in 1958-1962) were applied to the 1960 match population and were weighted for five years. Average annual Baltimore City rates (based on deaths in 1968-1972) were applied to the 1970 match population and were weighted for nine years.

Table 12

Age-and Time-Adjusted Mortality Rates per 100,000 for Lung Cancer

Males

Match I

Years	2303		2302		2404		Adjacent		South		Central		North	
	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	0	-	7	216.1	2	57.8	11	141.4	35	98.2	79	94.8	22	49.1
1960-62	4	166.8	5	107.5	6	119.2	12	99.7	44	84.9	114	89.9	38	55.3
1966-67	3	330.1	6	206.7	3	123.8	10	119.9	16	76.1	29	153.5	10	93.6
1968-69	3	180.0	3	104.4	7	241.2	5	61.8	30	141.4	10	52.7	16	166.4
1970-72	9	349.7	7	205.7	2	38.6	10	85.1	51	155.9	31	109.3	19	124.7
1973-74	6	512.5	2	61.7	3	106.1	9	104.9	30	139.3	19	100.0	13	126.3

Match II

	2301				Adjacent				Central				North			
	White		NW or B*		White		NW or B		White		NW or B		White		NW or B	
Year	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	2	140.8	0	-	1	46.7	0	-	4	223.0	3	144.6	5	83.0	3	42.4
1960-62	1	55.1	2	51.2	1	38.5	1	17.7	1	36.6	4	136.5	7	83.4	5	45.2
1966-67	1	61.0	0	-	10	322.0	7	160.4	1	35.1	1	87.1	5	149.0	0	-
1968-69	0	-	4	141.7	3	97.7	9	173.7	1	44.5	1	98.5	5	155.4	1	32.0
1970-72	4	181.7	7	191.7	6	122.2	4	65.7	3	81.0	3	258.8	3	57.4	3	67.2
1973-74	3	271.4	2	62.5	4	121.2	3	62.0	0	-	1	196.3	7	204.5	2	66.8

* 1958-59 and 1960-62 are non-white
All others are black.

Table 12A

Age-and Time-Adjusted Mortality Rates per 100,000 for Lung Cancer

Employee deaths removed

Males

Match I

Years	2303		2302		2404		Adjacent		South		Central		North	
	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	0	-	7	216.1	2	57.8	11	141.4	35	98.2	79	94.8	22	49.1
1960-62	4	166.8	5	107.5	5	104.1	12	99.7	44	84.9	114	89.9	38	55.3
1966-67	3	330.1	5	169.5	3	123.8	10	119.9	16	76.1	29	153.5	10	93.6
1968-69	2	94.6	3	104.4	7	241.2	5	61.8	30	141.4	10	52.7	16	166.4
1970-72	8	309.5	7	205.7	2	38.6	9	78.4	51	155.9	31	109.3	19	124.7
1973-74	6	512.5	2	61.7	3	106.1	9	104.9	30	139.3	19	100.0	13	126.3

Match II

	2301				Adjacent				Central				North			
	White		NW or B*		White		NW or B		White		NW or B		White		NW or B	
Year	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	2	140.8	0	-	1	46.7	0	-	4	223.0	3	144.6	5	83.0	3	42.4
1960-62	1	55.1	2	51.2	1	38.5	1	17.7	1	36.6	4	136.5	7	83.4	5	45.2
1966-67	1	61.0	0	-	10	322.0	7	160.4	1	35.1	1	87.1	5	149.0	0	-
1968-69	0	-	4	141.7	3	97.7	9	173.7	1	44.5	1	98.5	5	155.4	1	32.0
1970-72	4	181.7	7	191.7	6	122.2	4	65.7	3	81.0	3	258.8	3	57.4	3	67.2
1973-74	3	271.4	2	62.5	3	88.4	3	62.0	0	-	1	196.3	7	204.5	2	66.8

* 1958-59 and 1960-62 are non-white
All others are black.

Table 13

Age-and Time-Adjusted Mortality Rates per 100,000 for Lung Cancer

Females

Match I

Years	2303		2302		2404		Adjacent		South		Central		North	
	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	0	-	0	-	0	-	3	48.2	2	4.6	7	9.0	9	20.3
1960-62	0	-	0	-	0	-	3	32.6	6	15.2	11	10.7	6	10.3
1966-67	0	-	1	28.8	0	-	2	22.4	2	11.4	1	5.2	0	-
1968-69	1	123.5	0	-	0	-	0	-	6	21.9	7	34.2	1	6.4
1970-72	0	-	3	66.2	0	-	4	31.3	4	11.2	6	17.8	5	30.9
1973-74	0	-	0	-	1	27.3	3	31.7	6	23.0	5	26.4	2	15.5

Match II

	2301				Adjacent				Central				North			
	White		NW or B*		White		NW or B		White		NW or B		White		NW or B	
Years	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate	#	Rate
1958-59	0	-	0	-	0	-	0	-	1	142.9	0	-	0	-	0	-
1960-62	0	-	1	20.6	0	-	0	-	0	-	0	-	1	11.0	0	-
1966-67	0	-	0	-	0	-	0	-	0	-	0	-	2	50.6	0	-
1968-69	0	-	1	27.3	1	43.1	2	45.3	1	61.6	0	-	0	-	0	-
1970-72	1	36.3	0	-	1	27.6	2	22.5	0	-	1	56.5	1	16.5	0	-
1973-74	1	67.1	0	-	0	-	3	69.2	0	-	0	-	2	51.3	0	-

* 1958-59 and 1960-62 are non-white
All others are black.

Table 14

LIMITS ON SMR BASED ON A POISSON DISTRIBUTION

White Male Lung Cancer

Census Tract	Obs[a]	Exp[b]	SMR	LL	UL
2303*	9	2.14	4.21	1.93	7.98
0203*	9	4.00	2.25	1.03	4.27
2005*	15	6.10	2.46	1.38	4.06
2006	7	3.15	2.22	0.89	4.58
1205*	7	2.52	2.78	1.12	5.72
1207	8	3.92	2.04	0.88	4.02
2533	5	1.72	2.91	0.94	6.78
2553*	9	3.95	2.28	1.04	4.33
2611*	11	4.15	2.05	1.32	4.74
2636*	8	3.07	2.61	1.13	5.13
2653	2	0.98	2.05	0.25	7.37
2664	6	2.14	2.19	0.80	4.77
2737	2	0.98	2.05	0.25	7.37
2765	4	2.00	2.00	0.54	5.12

* 95% confidence limits does not include probability of 1.00 for the SMR

a Observed deaths in 1970-1972

b Expected deaths based on the average annual Baltimore City rate. Deaths in 1970-1972.

Table 15

Sampling Sites and Arsenic Content (ppm) by Depth of Soil

Summer 1976

Site	ppm As 1"	2"	3"	4"	Average of 1", 2", 4"	Site	ppm As 1"	2"	4"	Average of 1", 2", 4"
1-4	97	161	86	86	114.7	53-55	5.1	2.5	4.4	4.0
5-8	29	46	21	14	29.7	56-58	5.8	8.1	5.2	6.4
9-12	226	93	80	48	122.3	59	7.1			
13-16	87	101	59	69	85.7	60-62	4.0	1.1	3.0	2.7
17-18	14		7			63-65	1.5	7.5	4.0	4.3
19-20	6		4			66-68	695	55.2	24.6	258.3
21	8.9					69-71	9.0	5.4	4.0	6.1
22	8.9					72-74	8.9	9.7	5.3	8.0
23-25	76.9	102		18.7	65.9	75-77	8.9	12.5	5.4	8.9
26-28	96.5	67.8		56.5	73.6	78-80	3.2	7.7	5.2	5.4
29-31	65.1	137		15.2	72.4	81-83	11.5	12.7	11.4	11.9
32-34	163	85.7		22.1	90.3	84-86	2.6	3.1	4.6	3.4
35-37	41.0	100		46.8	62.6	87-89	3.0	2.1	19.0	8.0
38-40	63.8	126		102	97.3	90-92	5.0	4.6	2.8	4.1
41-43	34.6	38.8		54.7	42.7	93-95	4.5	2.8	4.5	3.9
44-46	63.7	73.1		13.2	50.0	96-98	6.2	4.4	2.8	4.5
47-49	25.7	45.6		17.8	29.7	99-101	11.0	7.5	6.5	8.3
50-52	6.1	3.5		16.9	8.8					

Spring 1977

Site	1"	2"	4"	Average of 1", 2", 4"	Site	1"	2"	4"	Average of 1", 2", 4"
1-3	17	5	6	9.3	19-21	12	34	12	19.3
4-6	9	13	4	8.7	22-24	6	29	144	59.7
7-9	10	8	2	6.7	25-27	4	9	20	11.0
10-12	41	56	60	52.3	28-30	90	173	15	92.7
13-15	4	22	19	15.0	Control 30-33	5	7	4	5.3
16-18	14	20	12	15.3					

Table 16

Non-Agreement of Certificate and Hospital Record of Cancer for Lung and Total

	Lung Cancer	Total
1958-67		
Total	99	414
Non-agreement	15	62
Percent	15.2	15.0
1968-74		
Total	119	356
Non-agreement	20	66
Percent	16.8	18.5

Table 17

Level of Agreement between Death Certificate Cancer Cause and Hospital Diagnosis

	Number	%	
Complete Agreement (4 digits in ICDA code)	555	75.0	80.7
Agreement to 3 digits	42	5.7	
Agreement to 2 digits	31	4.2	
Metastasis entered on D.C. as underlying	13	1.8	
Multiple cancers on D.C. Primary site not stated	41	5.5	
Other	18	2.4	
No cancer at autopsy or biopsy	20	2.7	
No records available	20	2.7	
Total	740		

Table 18

Smoking History in Lung Cancers from Index and Control Tracts by Sex

	Smoking		Non-Smoking		Unknown		Total
	No.	%	No.	%	No.	%	No.
Index Tracts							
Male	30	59	2	4	19	37	51
Female	6	67	3	33	0	0	9
Control Tracts							
Male	63	52	0	0	58	48	121
Female	5	22	0	0	18	78	23

Table 19

Cell Types of Lung Cancer by Census Tract

	Oat	Squamous	Adenocarcinoma	Epidermoid	Other	UK	Total
Tract 2303							
Male	2	8	-	1	1	4	16
Female	-	1	1	-	-	-	2
Other Index							
Male	3	18	2	1	1	8	33
Female	-	3	3	-	1	-	7
Control							
Male	12	57	16	4	3	18	110
Female	4	4	9	-	4	1	22

Table 20

Population for Sample and Follow-up

	1958-62	1966-67	1968-74
Index Population			
Total Deaths	148	72	153
Sample	148	72	153
Hospital request* to abstract	96	56	119
Control Populaton			
Total Deaths	2596	364	1030
Samole	591	258	566
Hospital request* to abstract	414	191	441

* Requests were submitted to all hospitals for those patients who died in one of the 11 cooperating hospitals. Some individuals died at home and no further confirmation could be obtained. Records were not obtained for all those requested.

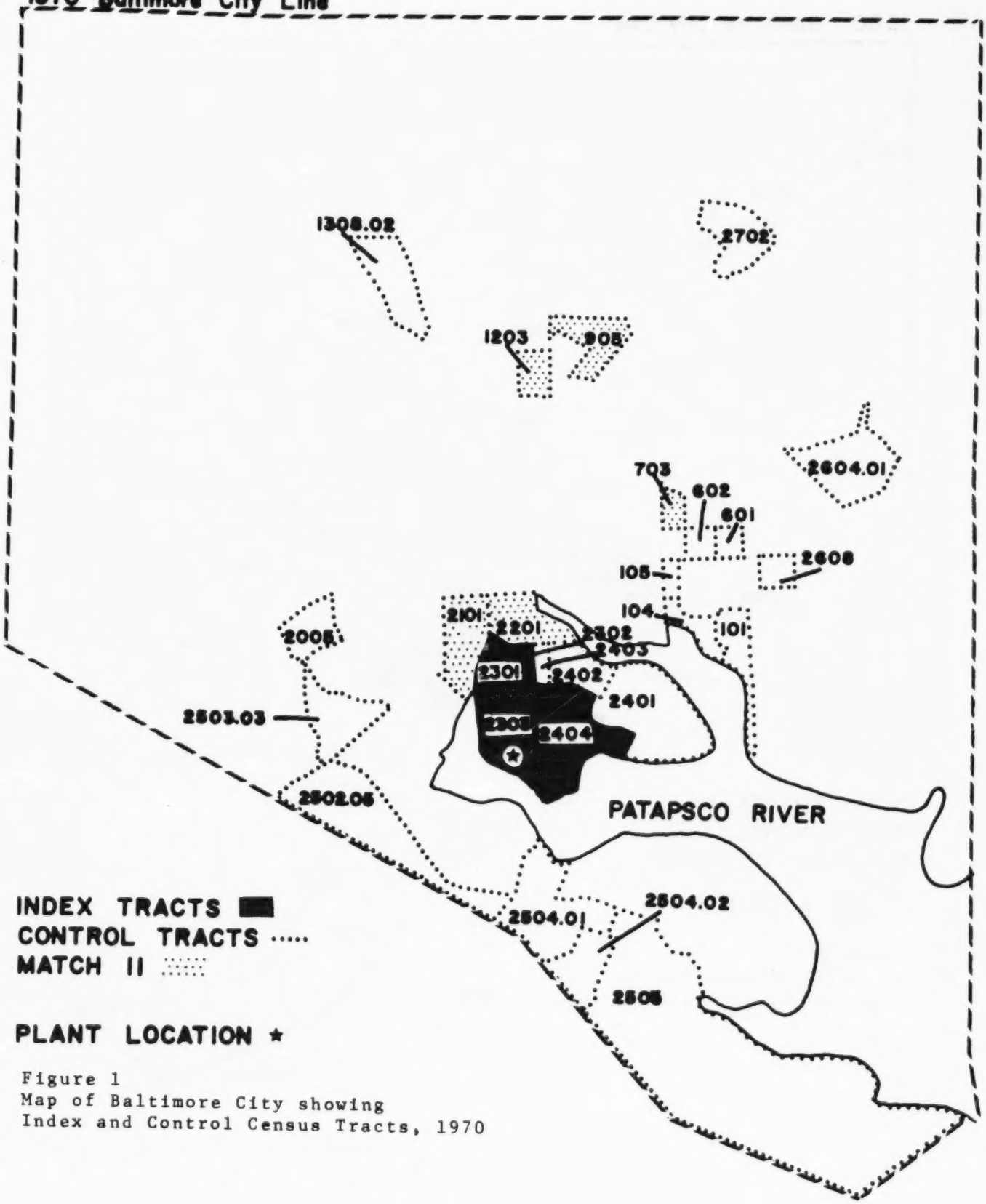

Figure 1
Map of Baltimore City showing
Index and Control Census Tracts, 1970

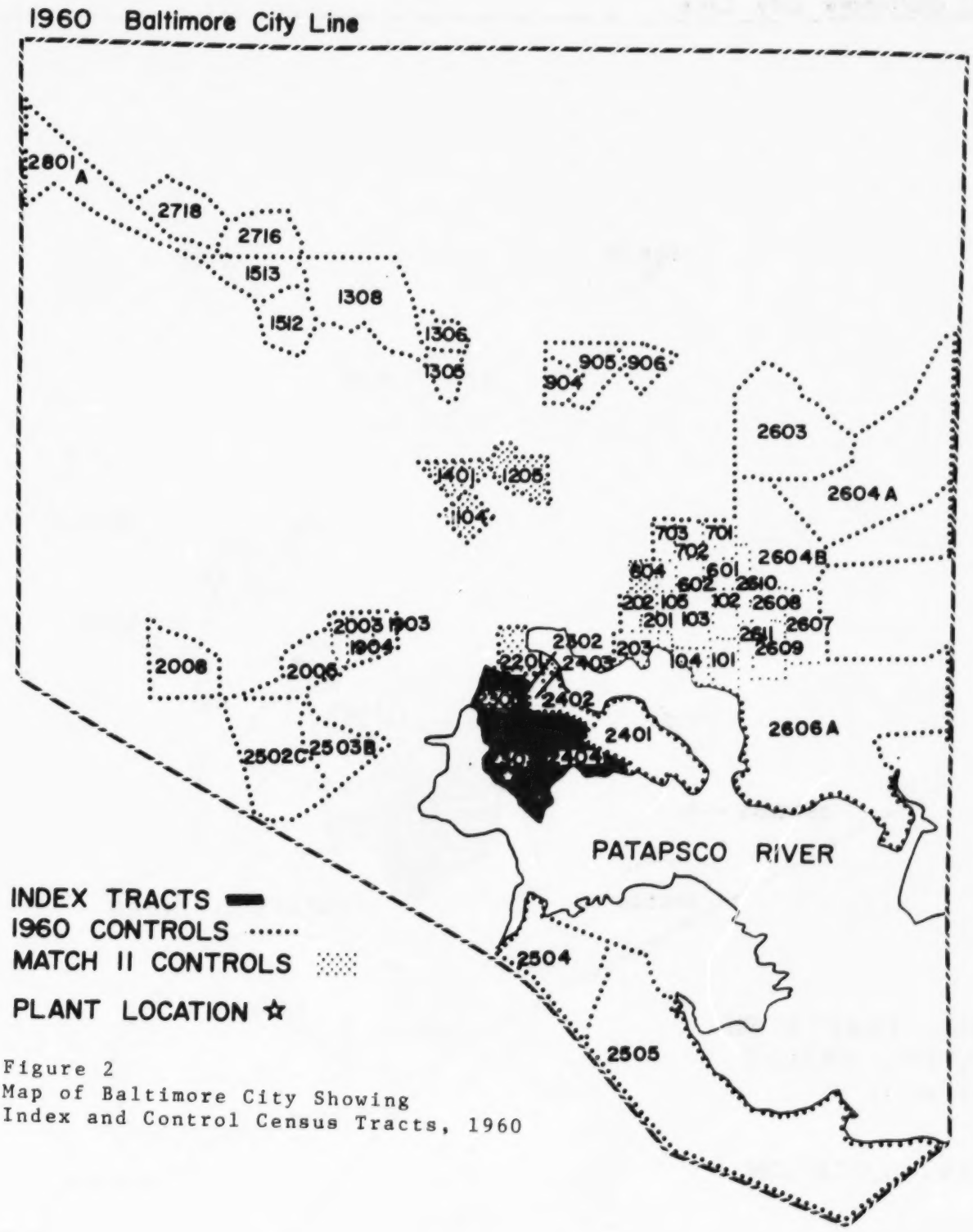

Figure 2
Map of Baltimore City Showing
Index and Control Census Tracts, 1960

Comparison of total cancer rates for census tract 2303 with controls. Age and time adjusted rates per 100,000 semi-logarithmic scale.

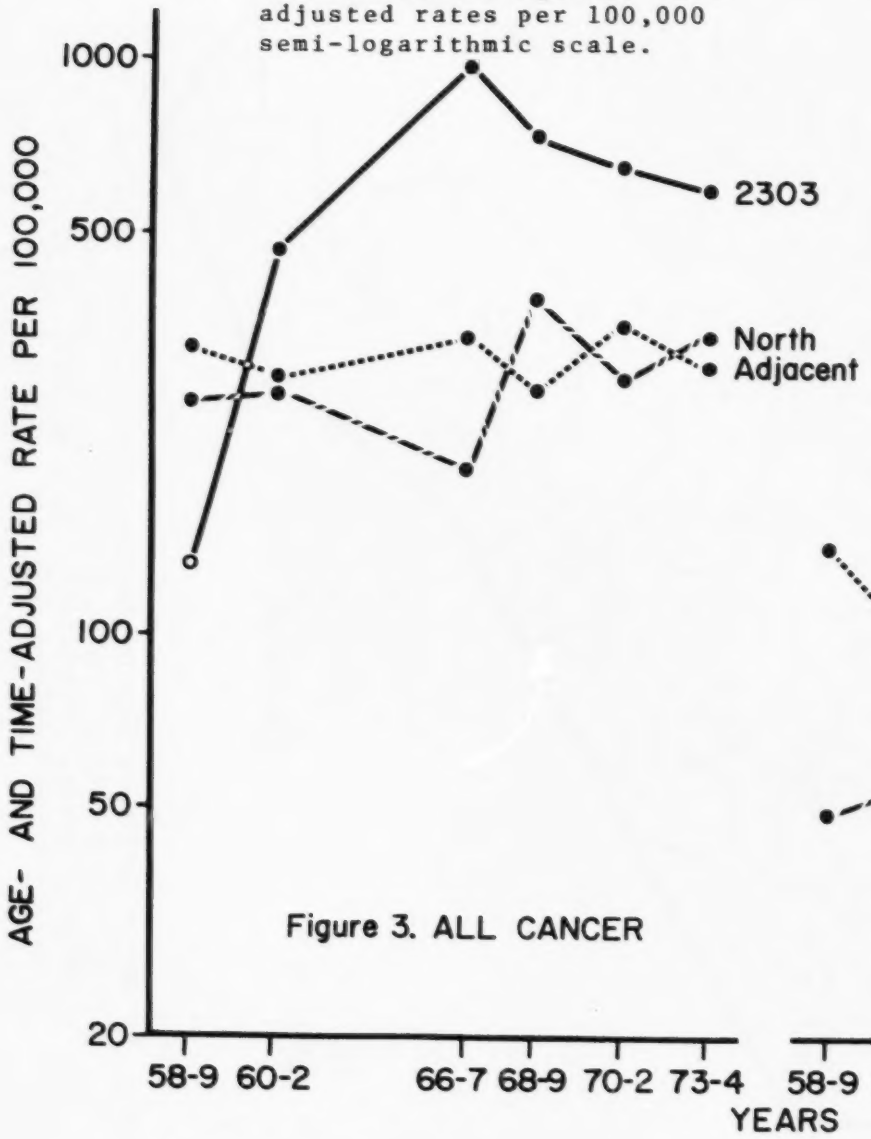

Figure 3. ALL CANCER

Comparison of Lung cancer rates for Census Tract 2303 with controls Age and time adjusted rates per 100,00 semi-logarithmic scale.

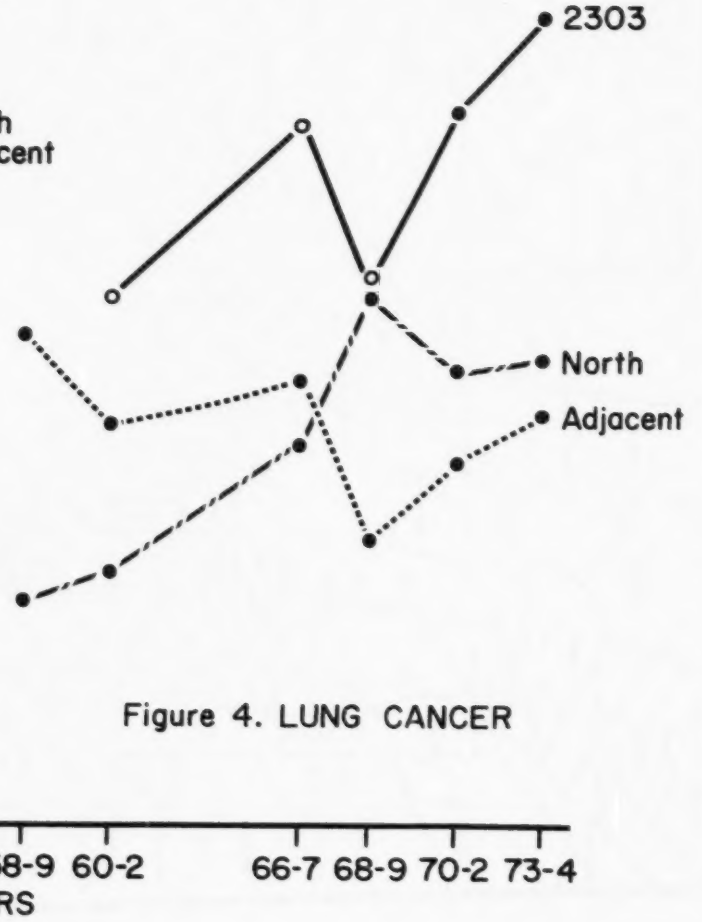

Figure 4. LUNG CANCER

o = less than 5 deaths

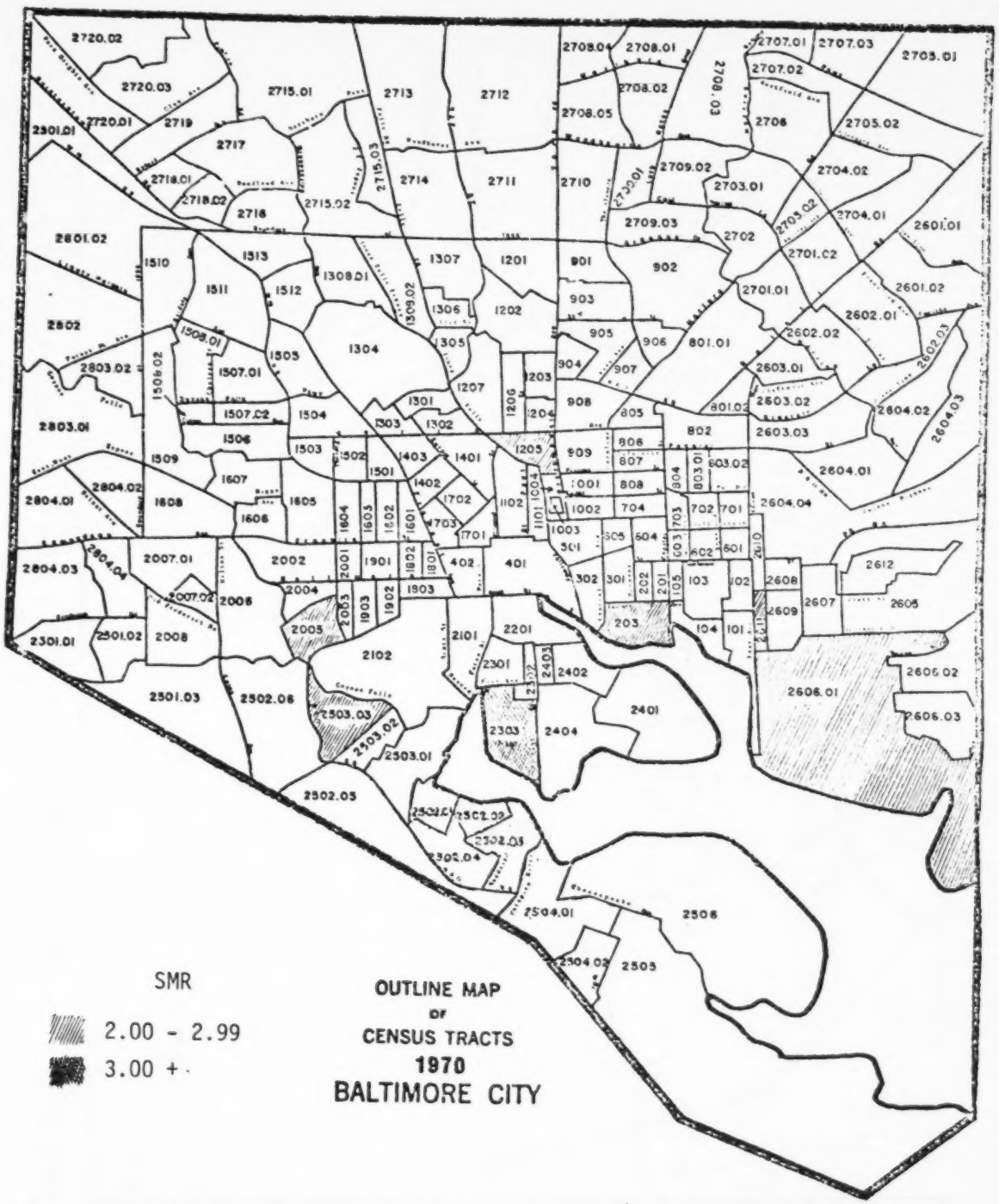

Fig. 5. Map of Baltimore City showing census tracts with statistically significant SMRs of 2 or more for white male lung cancer. (Observed deaths in 1970-1972. Expected deaths based on the average annual Baltimore City rate. Deaths in 1970-1972.)

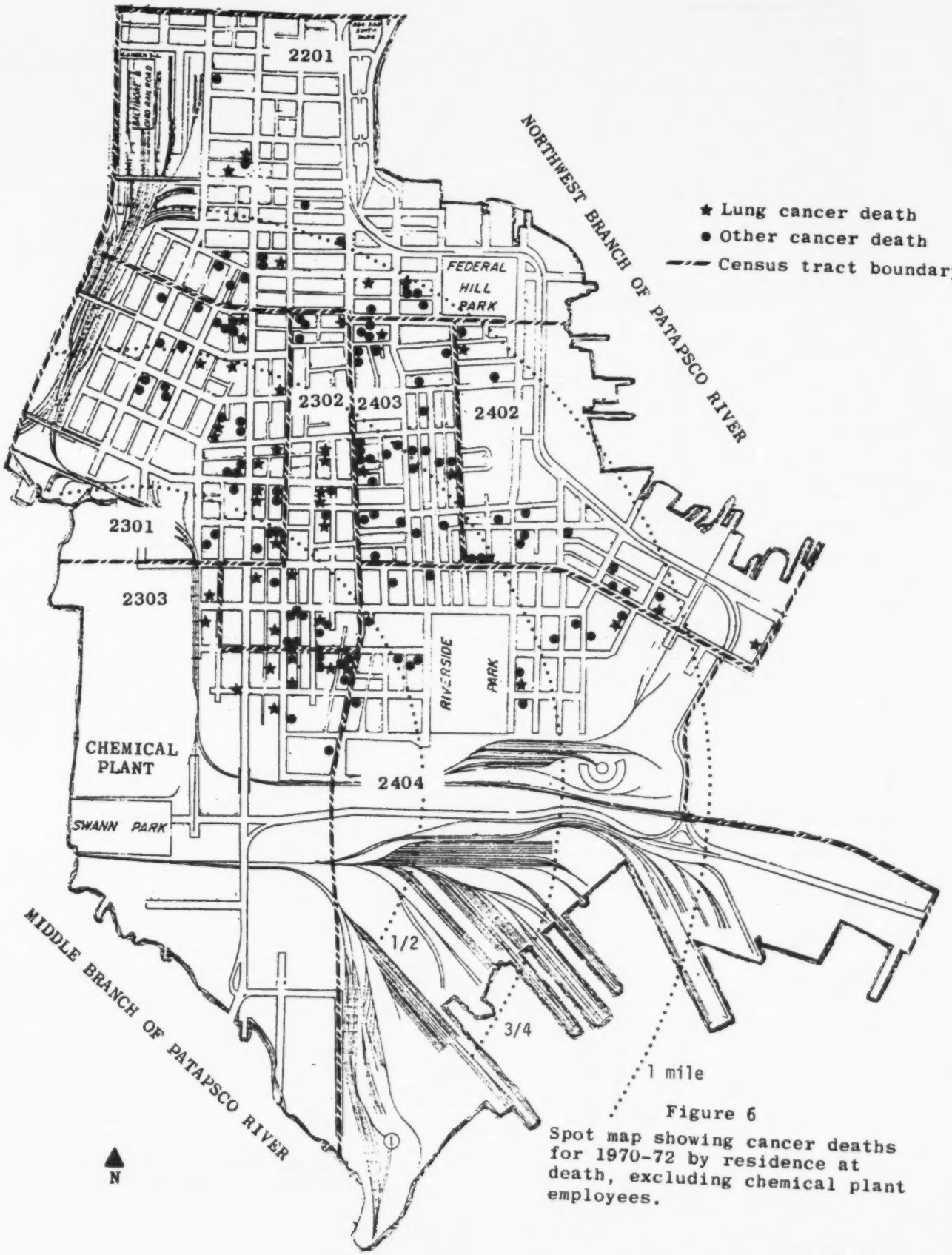

Figure 6

Spot map showing cancer deaths for 1970-72 by residence at death, excluding chemical plant employees.

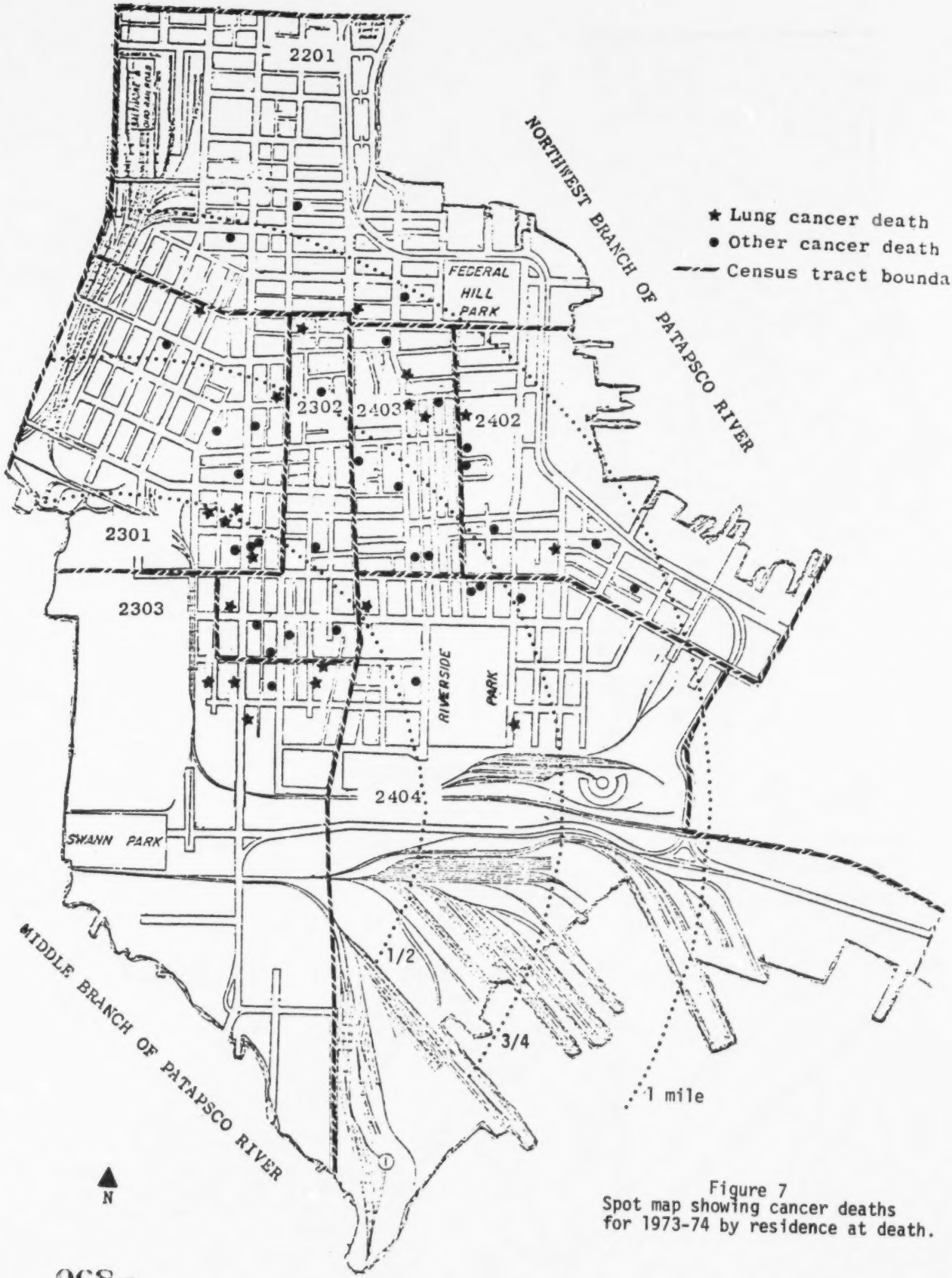

Figure 7
Spot map showing cancer deaths for 1973-74 by residence at death.

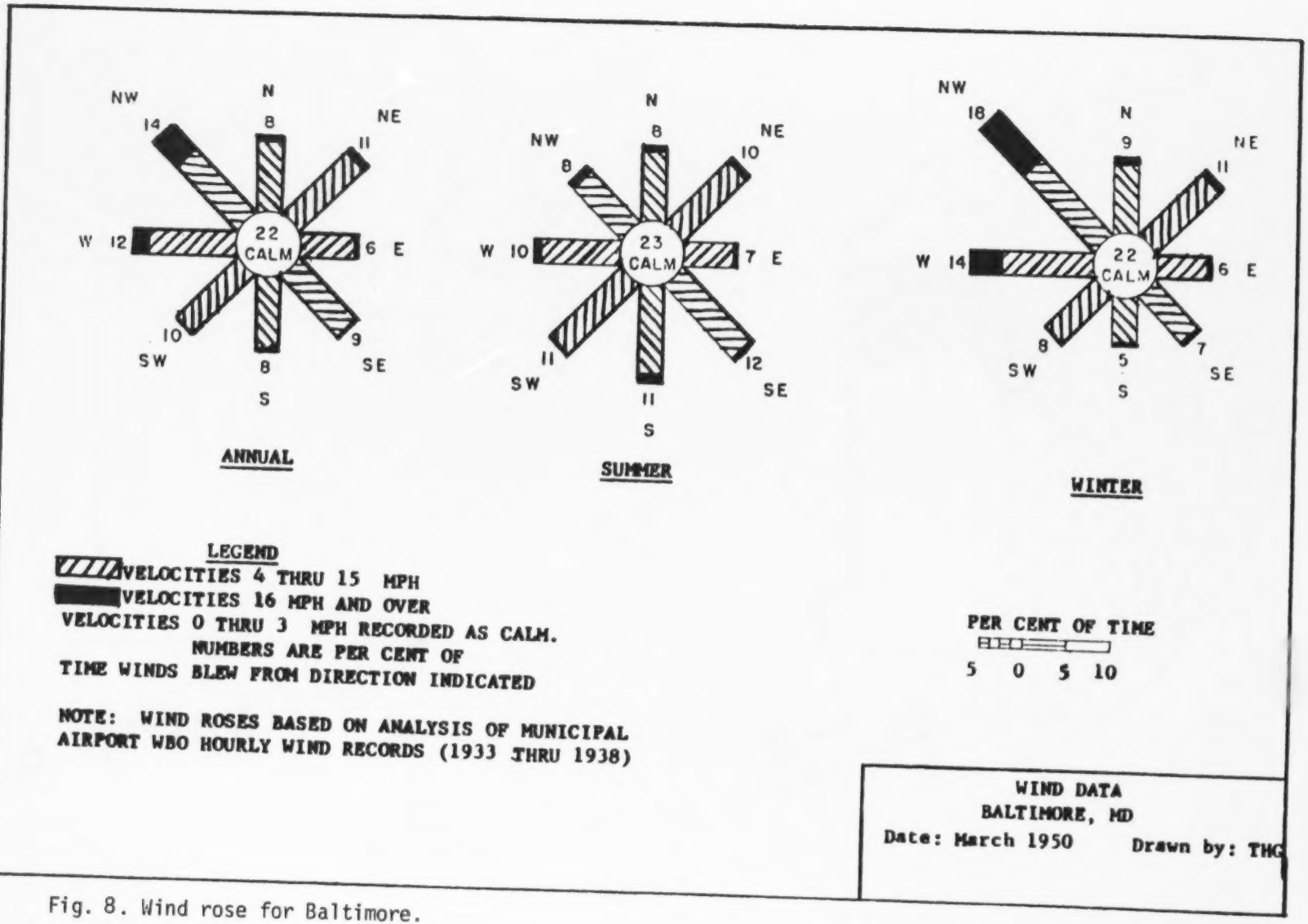

Fig. 8. Wind rose for Baltimore.

Fig. 9. Sampling Sites
Summer 1976 and Spring 1977

Fig.10. Arsenic level in soil - ppm
Highest value at each site
Summer 1976 and Spring 1977

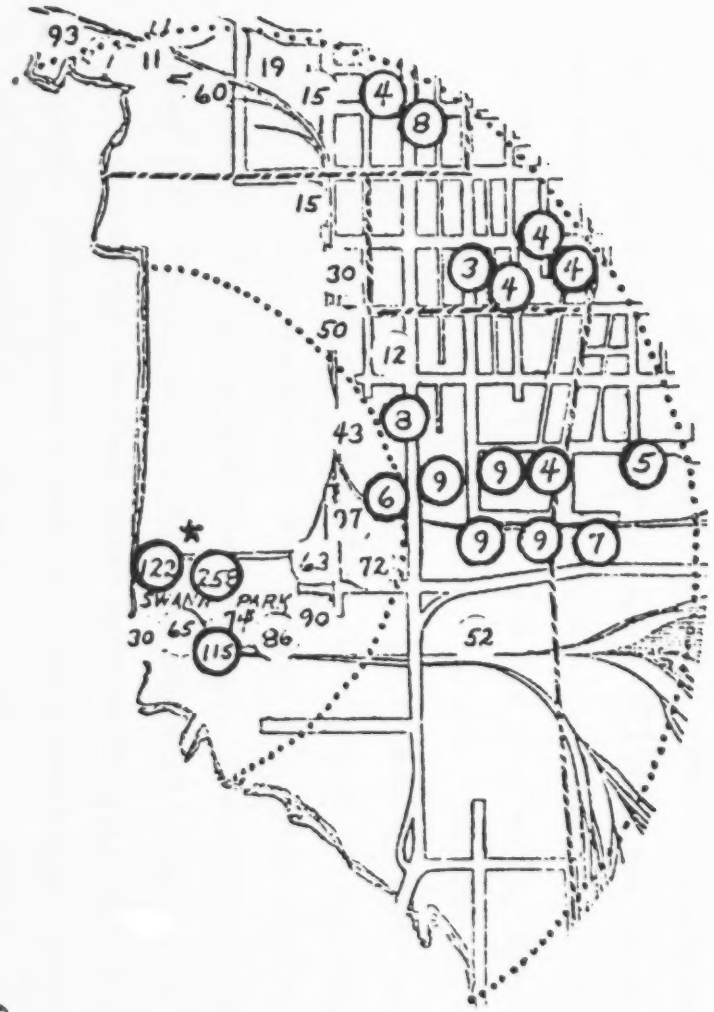

Fig. 11. Arsenic level in soil - ppm
Average of 1,2,4 inch samples
Summer 1976 and Spring 1977

METHOD OF SOIL SAMPLING

The original selection of sampling sites was determined both by distance from the plant and by direction from north through south coordinates. We intended to collect about half the samples within the 1/4 mile radius and 40 per cent at the next 1/4 mile distance with the remaining samples collected further out on the radii. Control samples would be taken from two parks nearby but at a distance greater than 1 mile from the plant. The field survey team had problems adhering to the sampling design since the sources of soil were limited in the area. We attempted to take samples near residences whenever possible as long as there were no obvious problems of tree-cover, water run-off, or redevelopment. For those few samples taken at private housing, the residents were interviewed concerning the use of herbicides or pesticides in the area and the sample was avoided if the soil had been treated. After collection of the original 101 samples taken at 35 sites under these directives and including additional samples in the park, a second set of samples was collected in a north and north-west direction to determine how far distant the high levels could be detected. Special emphasis was placed on sampling from the park which was adjacent to the plant. This park has a central grassed area which had been recently re-sodded. Surrounding the park was a dirt-track which had been undisturbed. Part of this path was adjacent to the

fence along the plant boundary and near the areas where railroad cars were filled. Another portion bordered on the water and the last was adjacent to railroad tracks.

Samples were collected at one, two and four inches at each location unless otherwise noted. A core sampler, with a 3.4 inch bore and marked at one inch intervals, was driven into the ground and samples were removed down to the appropriate depth marked. Since we had been informed that materials are often not uniformly distributed in soil, a one foot circle was marked off around a selected site and a set of samples was collected according to the described technique until the 30 ml. polyethylene sample bottle was filled with soil from the appropriate depth but from different core samples. This might require about four separate cores. Any debris such as rocks, trash, or grass was removed in a funnel prior to deposition in the bottle. Initially we had tested four sites using consecutive one inch samples down to a depth of four inches. We found that samples at three inches were usually close to those at four and thus it was elected to take the extreme depth and discard the three-inch level. Samples in control areas as indicated in table 1 were all obtained from two city parks, Riverside or Federal Hill.

All sampling was done with polyethylene equipment except for the corer and the stainless steel spatulas in

order to eliminate possible accidental arsenic contamination. The metal products used were reportedly free of arsenic and were the same throughout all sampling. Between each sample, the equipment was rinsed with tap water and cleaned finally in 100 mm ethylenediaminetetraacetic acid (EDTA) which was prepared in glass and stored in polyethylene containers.

The samples were mixed and renumbered before shipment tc the laboratory so that no pattern of numbers by depth or location could be detected by technicians running the samples. The analysis was done at Gulf South Research Institute under the direction of Ms. Mary M. McKown using the digestive procedure recommended by EPA. All particles 3 mm in size were removed. The sample was heated with HNO_3 and HC1 acids for 3 hours and analyzed by either conventional flame or flameless atomic absorption spectrophotometry depending on the level of arsenic.

METHOD OF VALIDATION OF HOSPITAL RECORDS AND PATHOLOGY OF CANCER CASES

Methods

The deaths for the total 14 years of study were included in the sample and stratified by control and index census tract. The sample for hospital record review included all deaths within the city for residents in the index tracts and one control tract which was to be used subsequently for the population survey. All city deaths were stratified by age, race, and sex and three time periods, 1958-62, 1966-67 and 1968-74. Four control deaths were selected randomly from each stratum for each index tract death within the same stratum. For the following analyses no attempt was made to expand the sample to the original population size. (See Table 20)

The hospital abstract form was designed to include information on the following variables:

1. Final diagnosis
2. Source of information for diagnosis
3. Symptoms of cancer
4. Associated medical conditions, especially cancer
5. Arsenic-associated symptoms

6. Personal characteristics as smoking and occupation
7. Family history, especially cancer
8. Laboratory studies including blood, X-rays, and EKG
9. Description of pathological specimens; operative or autopsy findings transcribed in detail.

Verification of the identification of the correct individual on the hospital record was done by name, birthdate, residence, and date of death.

Records were reviewed in eleven of the hospitals in Baltimore City. The other five city hospitals either refused to participate or still had not replied favorably to the request at the termination of the study. The non-cooperating hospitals were the smaller ones and did not limit substantially the number of records reviewed. It became apparent that problems arise with the scheme of validating patient cancer records through a review of data from the hospital listed on the death certificate. On several occasions, the patient had died at home and no hospital was listed on the certificate. In this case it was impossible to know where to begin to seek diagnostic information on the individual. An attempt to review registry files to identify these missing records proved to be inefficient. This information could only be provided by a survey. Another problem was that many individuals died at hospitals other than the ones in which the cancer was sparse. An attempt was made to follow these cases

through all hospital admissions in which the cancer may have been treated but this was not completed in all cases because of time, money, missing information, or non-inclusion of a hospital. In these cases we frequently cannot be sure of the cancer diagnosis. We did not examine the records of cancers or cancer deaths of city residents at hospitals in nearby counties. If the study were to be continued, all outlying hospitals should also be included.

All records were abstracted by two abstractors and were reviewed by a supervisor for editorial changes. All possible medical conditions found on record review were listed and coded by the same nosologist who codes all the death certificates. The celltypes were classified, in general, according to the Manual of Tumor Nomenclature and Coding. Since this coding scheme does not appropriately classify the cells of several tumors, especially those of non-solid origin, a revision of the scheme was made to include these cancers if we felt that their frequency was sufficient to warrant specific classification.